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ISCHEMIC HEART DISEASE; RESPONSE TO LEE

Dear Editor:

Dr. P. Lee questioned the reasons for a discrepancy of my reports in 1981 and in 1984 on husbands' smoking and ischemic heart disease risk in nonsmoking wives.

The 1981 report was based on a 14 year follow-up (n=400) and the 1984 report was based on a 16 year follow-up (n=494) of nonsmoking wives. The relative risks of ischemic heart disease when husbands were nonsmokers, exsmokers, or daily smokers of 1-19 cigarettes and 20 or more cigarettes were 1.00, 1.06, and 1.18 (trend p : 0.061 not significant) in the 14 year follow-up; and 1.00, 1.10, and 1.31 (trend p : 0.019 significant) in the 1984 report.

Table 1. Ischemic heart disease mortality in women by age group, by occupation, and by husbands' smoking habit (patient herself a nonsmoker).

Husband's occupation	Husband's age group	Husband's smoking habit: Nonsmoker		Exsmoker 1-19/day		20+/day		Total	
Agricultural worker	40-49	8	2,502	25	5,941	17	3,636	50	12,079
	50-59	15	3,497	27	6,812	27	3,514	69	13,823
	60-69	36	4,084	79	6,845	27	2,152	142	13,081
	70-	5	323	11	446	2	89	18	858
	Total	64	10,406	142	20,044	73	9,391	279	39,841
Other	40-49	5	3,727	15	9,093	15	7,128	35	19,948
	50-59	11	4,294	29	8,830	23	6,306	63	19,430
	60-69	29	3,036	46	5,598	20	2,499	95	11,133
	70-	9	432	8	619	5	137	22	1,188
	Total	54	11,489	98	24,140	63	16,070	215	51,699
The weighted point estimate of rate ratio and test-based 90% confidence limits		1.00		1.11 (1.33 / 0.92)		1.36 (1.68 / 1.09)		Mantel extension chi	2.539
Mantel-Haenszel chi		-		0.882		2.331		One tail p value	0.00916
One-tail p value		-		0.18889		0.00988			

Possible reasons would be (1) a longer follow-up period and more cases in the 1984 report than in the 1981 report, or (2) husbands' age and occupation were standardized for data in 1981, while data reported in 1984 was standardized by age only. However, the latter is definitely not the reason responsible for the discrepancy, as age-occupation standardized data in 1984 showed almost similar results, corresponding relative risks (r.rs) being 1.00, 1.11, and 1.36 (trend p : 0.009), respectively (Table 1). The results were also similar when standardized by wives' age, corresponding r.rs being 1.00, 1.09, and 1.34 (trend p : 0.019). Therefore, it should be concluded that the more cigarettes the husbands smoke, the higher the ischemic heart disease risk in nonsmoking wives.

In 1980-1981, r.rs of ischemic heart disease in nonsmoking wives were 1.00, 1.29, and 1.87 (trend p : 0.041) when husbands were nonsmokers, exsmokers/10-19 daily, and 20+ daily respectively. One may further consider as the possible reasons for this discrepancy the influence of the changing quality of side-stream smoke coming out of the ignited end of cigarettes in recent years due to the intensive chemical manipulation of the products (e.g., inclusion of tobacco additives) in order to lower tar and nicotine, to improve the flavor, etc. Also, the recent increase in fat consumption in Japan may interact on the risk of ischemic heart disease when exposed to passive smoking.

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REBUTTAL TO LEE/KATZENSTEIN COMMENTARY ON PASSIVE SMOKING RISK

Dear Editor:

Lee (1989) and Katzenstein (1989), in their commentary on Wells'(1988) paper, take issue not only with Wells' estimates of the magnitude of the mortality effect of passive smoking on nonsmokers, but question whether mortality occurs at all. Their arguments are based upon the alleged fragility of the epidemiological studies of passive smoking and disease; the potential for misclassification of subjects' disease, or exposure; possible confounding factors; and the lower doses of smoke to which nonsmokers are exposed relative to smokers.

Let us examine these issues one by one. Are nonsmokers exposed to such low doses of environmental tobacco smoke (ETS) that Wells' estimates of 46 000 nonsmokers' deaths per year from passive smoking are about "46 000 too high", as Lee asserts? Perhaps the most salient point to be considered: active smoking is a cause of more than one out of every six deaths in the U.S.A. every year (USSG 1989). Intentional exposure to tobacco smoke has been judged to cause coronary heart disease, atherosclerotic peripheral vascular disease, lung and laryngeal cancer, oral cancer, esophageal cancer, chronic obstructive pulmonary disease, chronic bronchitis, intrauterine growth retardation, and low birthweight babies. In addition, probable causality has also been established for unsuccessful pregnancies, increased infant mortality, and peptic ulcer disease, as well as cancers of the bladder, pancreas, and kidney, and associations have been reported for cancer of the stomach (USSG 1989). Hardly an organ system of the human body remains undiseased upon exposure to tobacco smoke. To argue, as do Lee and Katzenstein, that the diseases of smoking are not even plausible in nonsmokers does not give us confidence in their deductive abilities. To be sure, it is possible that thresholds for effect may exist for one or more of the diseases of smoking, but neither Lee nor Katzenstein present any evidence whatsoever that such low dose thresholds exist, let alone that all nonsmokers have exposures and susceptibilities which place them within an adequate margin of safety below such thresholds.

Are the epidemiological studies of passive smoking and lung cancer really all to be explained by misclassification of smokers as nonsmokers as Lee has proposed? Nonsmokers who report no passive smoking nevertheless possess levels of nicotine and cotinine in body fluids which are significant fractions of those who report a lot of exposure. For